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Objective: Studies in high-income countries have shown that longer duration of breastfeeding in infancy is associated with a lower risk of later hypertension, type 2 diabetes and obesity. There is controversy as to whether this is causal or reflects confounding. Few studies have related age of introduction of complementary foods in infancy to later outcomes. Our objective was to test the hypothesis that longer duration of breastfeeding and later introduction of complementary foods are protective against adult hypertension, diabetes and overweight/obesity.

Methods: Data were pooled from 10,912 men and women aged 15–41 years, from 5 birth cohorts in low- or middle-income countries (Brazil, Guatemala, India, Philippines, South Africa) comprising the COHORTS collaboration (Consortium on Health Orientated Research in Transitional Societies). Exposure measures were 1) ‘ever’ versus ‘never’ breastfed; 2) total duration of breastfeeding (9 categories from no breastfeeding to breastfed for more than 24 months) and 3) age at starting complementary foods (6 categories from 0–3 months to >18 months). Outcomes were adult blood pressure, hypertension/pre-hypertension, plasma glucose concentration, diabetes mellitus, impaired fasting glucose, skinfolds, waist circumference, percentage body fat, and overweight/obesity. Analyses were adjusted for maternal socio-economic status, education, age, smoking, race and urban/rural residence, and infant birthweight. Each cohort study was approved by an appropriate institutional ethics committee and participants gave informed consent.

Results: There were no differences in outcomes between adults who were ever breastfed compared with those who were never breastfed. Associations between duration of breastfeeding and adult systolic blood pressure and prevalence of hypertension were U-shaped; however these were weak and inconsistent between cohorts. Duration of breastfeeding was not associated with adult diabetes or adiposity. Participants who started complementary foods later in infancy had lower adult BMI, waist circumference and subscapular skinfold thickness ($p < 0.01$ for all). BMI changed by -0.19 kg/m^2 (95% CI -0.37 to -0.005) and waist circumference by -0.45 cm (95% CI -0.88 to -0.02) per 3-month increase in age at introduction of complementary foods between birth and 9 months. These associations were not significant after adjusting for 2-year weight.

Conclusions: There was no evidence that a longer duration of breastfeeding protects against adult hypertension, glucose intolerance or overweight/adiposity in these populations. Delaying the introduction of complementary foods until 6 months, as recommended by WHO, may reduce the risk of adult overweight/adiposity. This may be mediated by lower infant weight gain.

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Does childhood nutrition contribute to sex differences in risk factors for ischaemic heart disease in a developing population?: The Guangzhou Biobank Cohort Study

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Objective: A male epidemic of ischemic heart disease (IHD) emerges with economic development. We have previously hypothesized, based on physiological and epidemiological evidence, that this epidemic is due to nutritionally driven levels of pubertal sex-steroids, which generate a life long more atherogenic body shape and lipid profile in boys but not girls, without any sex-specific effects on glucose metabolism, which is in turn more strongly related to the somatotrophic axis. Here we tested this hypothesis by examining the association of childhood (≤ 18 years) meat eating with these IHD risk factors in older adults from a developing Chinese population.

Methods: Multivariable linear regression was used in a cross-sectional study of 19,418 Chinese older (≥ 50 years) men and women from the Guangzhou Biobank Cohort Study (phases 2 and 3) to assess the adjusted associations of childhood meat eating with waist hip ratio, HDL-cholesterol and fasting plasma glucose.

Results: Childhood meat eating had sex-specific associations with waist-hip ratio but not fasting glucose. Childhood daily meat eating compared to less than weekly meat eating was associated with higher waist hip ratio (0.007 [95% confidence interval 0.0002 to 0.01]) in men but not women, adjusted for age, life course socio-economic position and current lifestyle.

Conclusion: This study adds to a growing body of evidence suggesting that puberty may be a key developmental window when sexual dimorphism in IHD risk emerges. The male epidemic of premature IHD and sexual divergence in IHD rates which occur with economic development may be nutritionally driven in childhood or adolescence, with corresponding implications for men in the developing world currently experiencing the epidemiological and associated

nutrition transition during early life. In elucidating the developmental origins of non-communicable chronic diseases more attention should be focused on socio-historical context and the hitherto overlooked role of puberty. Acknowledgements: The University of Hong Kong (HKSAR), Guangzhou Public Health Bureau (China), Guangzhou Science and Technology Bureau (China), The University of Birmingham (UK).

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The Pro12Ala polymorphism of the PPAR- γ 2 gene interacts with low birth weight in increasing the risk for myocardial infarction

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Objective: The PPAR- γ 2 gene regulates adipocyte differentiation and plays an important role in glucose, lipid and energy metabolism. The substitution of a proline by an alanine in codon 12 of the PPAR γ 2 interacts with size at birth in its effects on insulin sensitivity, lipid metabolism, blood pressure and antihypertensive medication in adulthood. Our objective was to investigate the effects of this polymorphism and birth weight (BW) on insulin resistance, blood lipids levels and blood pressure at age 50 and on the incidence of myocardial infarction (MI) at age 50–85 in a cohort of Swedish men.

Methods: 2322 men comprise the Uppsala Longitudinal Study of Adult Men (ULSAM). Of these, we had complete data on birth weight from archives and genotyped data for 674 men from clinical investigations at age 71 with 154 cases of incident MI recorded in routine registers over 36 years of follow-up. The Pro12Ala polymorphism (rs1801282) was genotyped using a 1536-plex Golden Gate Assay and the Bead Station genotyping system from Illumina. The data was analysed by linear and Cox regression in STATA 10, with formal tests for interaction with birth weight categories. All analyses were adjusted for BMI and age. Appropriate institutional ethics committee clearance and participants' informed consent were obtained.

Results: 158 (23%) of the 675 subjects carried either the Pro12Ala or Ala12Ala polymorphism. These subjects are compared to those carrying the wild type Pro12Pro variant in all comparative analysis. We observed statistically significant differences between the SNP variant and the wild type in mean levels of glucose measured at 0 min and 60 min after an intravenous glucose tolerance test in the

lowest birth weight tertile (<3290g), but no differences in lipid levels or blood pressure. The effects of the Pro12Ala and Ala12Ala polymorphism on MI also depended on the birth weight of the subjects. Subjects with the Pro12Ala/Ala12Ala polymorphism in the lowest birth weight tertile had an increased risk for MI (hazard ratio, HR = 1.93, $p = 0.03$ adjusted for BMI and age) while no significant effect was seen in subjects in the middle and highest birth weight tertiles. The tests for interaction between birth weight and the polymorphism were not statistically significant.

Conclusions: The Pro12Ala and Ala12Ala genotype is associated with lower blood glucose levels but increases the risk for MI in subjects with a low birth weight (<3290g). These results for MI are in contradiction to previous reports of this particular genotype providing a protective effect on insulin resistance and type 2 diabetes in subjects born with low birth weight. Support source: Wallenberg Consortium North and Swedish Council for Working life and Social Research.

Table. Differences in mean glucose levels and hazard ratios (HR) for MI in men with the Pro12Ala/Ala12Ala polymorphism compared to the Pro12Pro wild type, stratified by birth weight.

Birth weight (g) Tertiles	Subjects (n = 674)	Glucose 0 min	P	Glucose 60 min	P	MI cases	HR	95% CI
≤3290	167	-.2178	0.03*	-1.1863	0.03*	47	1.93	1.06–3.52
3291–3959	337	-.1863	0.06	-.7201	0.08	76	1.25	0.75–2.10
≥3960	170	-.1128	0.34	0.2525	0.65	31	0.80	0.32–2.00

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Analysing longitudinal measurements of size early in life as predictors of later outcomes

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Objective: In the “Cohorts” collaboration a recurring analytical issue is how to measure the association between an outcome measured in adult life, for example blood pressure, and a sequence of height, weight and body mass index measurements made early in life.

Methods: We illustrate the issues using data on glucose tolerance among women in the New Delhi study. We discuss regression models, focussing especially on conditional growth models. Conditional growth models divide the age range of size measurements into a sequence of intervals in each of which growth is measured as the difference between the size observed at the end of the interval and the size predicted from